Abstract

Drosophila melanogaster possesses a single adenosine receptor called dAdoR, whose functions in living processes are still unknown. By overexpressing or silencing *dAdoR* in photoreceptors, neurons, or glial cells, I examined fruit flies' survival, fitness, sleep, and locomotor activity.

In the survival assay, I observed that *dAdoR* overexpression causes early deaths in younger flies (1-10 days old), while silencing prevents early deaths and stabilizes median survival. However, I observed that experimental flies with silencing of this gene show a reduced overall lifespan. I found that overexpression of adenosine receptors in neurons and glial cells improves the fitness of older flies (60 days old), while silencing deteriorates their fitness.

In sleep experiments, I observed that *dAdoR* overexpression increases day sleep (*siesta*) in photoreceptors and night sleep in glial cells, while in all neurons, it increases total sleep. However, silencing of *dAdoR* did not show significant changes in sleep.

To study synaptic mechanism of behavioural changes, I examined the level of presynaptic protein Bruchpilot (BRP), and its daily pattern in the fly's first optic neuropil (lamina), after *dAdoR* silencing in photoreceptors or glial cells. I confirmed that the BRP protein oscillates in the tetrad synapses and shows significant changes at the beginning (ZT1) of the day and in the middle of the night (ZT16). During the evening peak of locomotor activity (ZT13), the protein level was highest in both experimental and control flies. However, after silencing of *dAdoR* in glial cells, I observed that BRP level changes only at the beginning and the middle of the day (ZT1 and ZT4).

In the final part of my thesis, I studied the possible effects of caffeine on functioning of adenosine receptors in sleep regulation, ageing, and behaviour. In wild-type flies I found that caffeine affects more strongly female flies, and it influences *siesta*. Also, caffeine is unable to disrupt the circadian clock when *dAdoR* is overexpressed or silenced (in all neurons, *pdf*-expressing clock neurons, *tim*-expressing neurons or *th*-expressing dopaminergic neurons). I observed that caffeine treatment decreases *siesta* when *dAdoR* is overexpressed in all neurons, *tim*-expressing neurons or in *th*-expressing dopaminergic neurons. In turn, *dAdoR* silencing increases *siesta.* This shows that adenosine receptors are involved in the regulation of *siesta*.

Keywords: adenosine, locomotor activity, sleep, synaptic plasticity, circadian rhythms, caffeine.